of a heart attack in the forties. The patient himself, though otherwise healthy, had always been somewhat short of breath on going uphill.

TREATMENT.

I know of no drug but morphine which will relieve the pain of coronary thrombosis, and it must be given freely. A quarter of a grain is generally insufficient, and my own practice, in severe cases, is to give an initial dose of half a grain hypodermically. When the blood pressure is low active stimulation is indicated, and apart from the relief of pain the treatment resolves itself into that of "shock"—keeping the patient warm, raising the foot of the bed, giving brandy or other stimulants by mouth, and camphor subcutaneously. I have tried ephedrine in some cases, but am not fully convinced of its value. Measures such as these may tide over the danger period. Let me repeat that, if there is one group of drugs more strongly contraindicated than any other in these cases, it is the nitrites. Continuous oxygen inhalation may be helpful when oedema of the lungs is present, though it cannot be expected to do good when the cyanosis is attributable solely to a diminished cardiac output.

The rapid heart action associated with auricular fibrillation, or paroxysmal tachycardia, which sometimes complicates coronary thrombosis, may in itself endanger life. In the former digitalis given by the massive dose method of Eggleston or intravenous strophanthin is called for; while the only drug which seems to have any effect in arresting the paroxysms of tachycardia is quinidine; six grains may safely be given, and repeated, if necessary, at two-hourly intervals for five doses.

To the importance of complete rest I have already referred. After a severe attack of coronary thrombosis the patient should not be allowed even to sit up in bed for two or three weeks.

CONCLUSION.

In the short time at my disposal I have only attempted to deal with the more important practical points in connexion with coronary occlusion. I have endeavoured to show that the study of coronary thrombosis is not confined to the post-mortem room. It is a definite clinical entity, which, in a large percentage of cases, can be recognized with certainty during life. Its recognition is important, because many of the patients survive, and a few are sufficiently restored to health to resume their former occupations. Further, a correct diagnosis is of vital importance, not merely in framing the prognosis, but also in determining the treatment. An acute coronary occlusion may closely simulate an abdominal crisis, and if unrecognized may be submitted to laparotomy, which, in view of the cardiac lesion, is almost certain to prove fatal. Other cases are mistaken for ordinary angina and treated with nitrites—a dangerous procedure in view of the low blood pressure.

The crescendo character of the pain, its duration, the fact that it is not relieved by rest and that the attack frequently occurs when the patient is resting and is in no way attributable to exertion, are important. The fall in blood pressure, the shock, prostration, and vomiting which generally accompany the pain, serve to distinguish it from true angina, as do the feeble first heart sound, the gallop rhythm, the rapid pulse, and the pericardial friction which are sometimes present. I need hardly say that one must not expect to find all these signs in every case, but there is generally ample evidence, even without the electrocardiograph, which, if it be available, will often clinch the diagnosis.

The aspect of prognosis is so difficult that I almost hesitate to refer to it, though my own experience would lead me to believe that even the most desperate cases may recover, especially when the patient is under 50 years of age.

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THE CORONARY ARTERIES IN A CASE OF FAMILIAL LIABILITY TO SUDDEN DEATH.*

BY

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WE publish the following record in the hope of eliciting from others, and especially from family practitioners of long experience, notes of similar cases.

In 1924 Carey Coombs and J. J. S. Lucas described to the Association of Physicians a family of whom the father and two sons had died with great suddenness. They had examined the heart of one of these sons, a man aged 32, who had been taken ill while playing football and had died within an hour or so; it was found to present macroscopical changes apparently of an atheromatous type in the coronary arteries. These changes had not been histologically examined as the specimen had been lost. They also gave particulars of a third son who had been seen by them for symptoms suggesting angina pectoris.

This man was first seen in 1923. The history was that in 1918, during exercise, he complained of an uncomfortable tight feeling across the chest. This passed off, and it was not until 1923 that

FIG. 1.

the attacks became at all frequent. He then stated that for the previous ten months the attacks had been getting more frequent, and were brought on more and more readily, till in the latter part of the year he could not run ten yards without experiencing a tight gripping sensation in the chest. He never became dyspnoeic because the feeling came on before he had time to get out of breath. It never occurred at night, but was more easily brought on after a meal.

In May, 1925, one of us (C. E. K. H.) was asked to take an electro-cardiogram. (See Fig. 1.) The following is a genealogical tree of the family.

Heavy type indicates affected members.

In October, 1927, the patient was sent to see one of us (C. E. K. H.). He stated that the attacks were now associated with pain. A sudden tight feeling over the sternum was associated with an acute pain in the same region which was quite stationary,

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FIG. 3.—Radiograph of the injected heart, showing obstructions in the coronary circulation and the increased vascularity of the left ventricle.

FIG. 4.—Radiograph of a normal injected heart of the same age, showing the "age change" occurring normally, for comparison.

FIG. 5.—Radiograph of the injected heart opened out, showing obstructions in the coronary circulation and the increased vascularity of the left ventricle.

FIG. 6.—Low power photomicrograph of one of the larger branches of a coronary artery, showing (1) the injection mass; (2) the thickened intima.

FIG. 7.—Higher power view of same, showing (1) the injection mass; (2) the thickened intima; (3) the media; (4) the adventitia.

and he felt bound to stand still for a few minutes, after which he was able to carry on again. The pain had never occurred at night, but was definitely produced more easily after food. The attacks had gradually got more frequent, though not more severe, and the exercise of quiet walking was sufficient to bring one on. Latterly he had had two or three attacks a day.

On physical examination the heart was not enlarged. An x-ray outline was taken, and this showed a heart of normal shape, but the aortic shadow was wider than normal. The heart sounds were clear, though the first sound was rather high-pitched; the aortic second sound was not accentuated. The arteries were not thickened, and the blood pressure was 120/70. The urine was normal, the Wassermann reaction negative, and no abnormal signs were present. Another electro-cardiogram was taken (Fig. 2), and this showed a marked difference in the "T" complexes.

FIG. 2.

Whereas in the former one all the T's were positive, in this second one T2 is barely positive and T3 is definitely negative.

A month after this examination he fell down dead when getting into a tram-car.

Post-mortem Examination.

The post-mortem examination was performed at the Bristol Royal Infirmary by Dr. A. D. Fraser, and by his kindness one of us (C. B. P.) was able to examine certain organs in detail.

The subject was a well-nourished male about 40, and there were no external evidences of trauma or of disease. The lungs were normal in appearance, as also were the abdominal organs, with the exception of the aorta and kidneys. The aorta in its thoracic part showed slight atheroma, but the atheromatous change was very marked in the abdominal portion. There were several atheromatous ulcers, and in one or two places calcification had occurred. On section the aortic lesions show pronounced intimal thickening. In most places the media is the seat of replacement fibrosis. The intimal lesions in some parts show extensive fatty degeneration, and around the fat deposits are a certain number of mononuclear cells with a foamy cytoplasm, apparently engaged in phagocytosis of the fat. This is the only cellular reaction to be seen in the lesion.

The heart was somewhat enlarged, and on palpation the coronary arteries were definitely hard and thickened. It was injected with a barium gelatin emulsion, through the coronary arteries, according to the technique described by Gross,¹ and x-ray photographs were taken.

The skiagram of the injected heart shows intense vascularization of the left ventricle. This increase in vascularity of the left side compared with the right corresponds to a very extreme degree of the "age change" described by Gross, although in this case it is much more marked than one would expect at twice the age. At the age of 40 the increase of the blood supply of the left ventricle over the right is normally only just becoming obvious. In both the right and left coronary areas there are obstructions to the circulation.

All four valves were normal in appearance. On microscopical examination the myocardium shows areas of dense fibrosis. The coronary arteries are in many places almost completely occluded by massive intimal thickening, similar in type to that occurring in the aorta. In places this intimal thickening has been vascularized by new capillaries. Both internal and external elastic laminae are, in places, broken up, and the media shows some increase in the amount of fibrous tissue it contains. There appears to be some increase in the adventitial layer of fibrous tissue.

The kidneys were normal in size and appearance. There was very little abnormality in the small renal vessels; the renal artery shows medial fibrosis, but no intimal change.

The cause of death would appear to have been impairment of the coronary circulation due to degenerative changes in the coronary arteries. The pathological process corresponds with that of a premature senility of the arterial tree, with a particular incidence on the coronary arteries. There is no evidence of any infective factor. The pathology of the coronary artery lesions resembles that in the case described by Perla and Seligman,² except that

in their case the lesion was by no means confined to the coronary arteries.

We may also refer to a recent paper from this laboratory in which Hughes and Perry³ report the case of an infant, aged 7 weeks, who died suddenly, and was found at the necropsy to exhibit advanced degenerative changes in the coronary arteries.

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THE USE OF SURGERY IN PULMONARY TUBERCULOSIS.*

BY

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IN the treatment of pulmonary tuberculosis, as in that of all other diseases, the simplest methods, carrying the least risk, must be tried first and continued as long as they show a possibility of succeeding. The simplest treatment for many patients with pulmonary tuberculosis is sanatorium treatment, being generally without risk. In a few cases, however, there is the danger that haemorrhage may curtail life before healing takes place under sanatorium regime. Again, sanatorium treatment may be inadequate on account of the acuteness of the disease, weak powers of resistance, recurrence of infection, or because the character of the changes, whether the result of the tubercle bacilli, or of the secondary infection, or of the resulting fibrosis, is such that healing cannot occur without the application of radical additional aid.

In this article it is proposed to consider the problems of how to diminish the relative virulence of the tubercle bacilli to the resistance of the individual, and of how to reduce the anxieties and dangers of haemorrhages: how, further, the healing processes and the eradication of the infectivity of the individual can be hastened when the time factor is of importance: and finally, how we can overcome the persistent infections of single large or of multiple small cavities in the parenchyma or in the dilatations of the bronchi.

Where one lung is involved the object is achieved if collapse can be readily secured, but this is not always possible. If the second lung is implicated, the radical treatment of the more seriously affected side may have to be greatly modified, and may even be impracticable.

The simplest, but not always the easiest, method of collapse is that of artificial pneumothorax. The purpose here, however, is to discuss those surgical methods which may be accessory or alternative to artificial pneumothorax—namely, hemidiaphragmatic paralysis by phrenic evulsion, thoracoplasty, multiple neurectomy, and extrapleural pneumolysis. If it were always possible to get a perfect collapse with artificial pneumothorax, and if there were no complications to this method of treatment, there would be less need for these other operations. What often indicates the necessity for the operations of thoracoplasty or phrenic evulsion are the failures to obtain complete collapse by artificial pneumothorax, even with the aid of the cautery for dividing adhesions, and the various complications of pneumothorax.

The Operation of Evulsion of the Phrenic Nerve.

This operation consists in exposing the nerve in the neck, dividing it, and then, by traction on the distal end, drawing out the intrathoracic part of the nerve. In a case of complete evulsion the terminal filaments will be torn out from the diaphragm. It is not advisable, however, in certain cases, such as in those in which the nerve is caught up by thickened pleura, to attempt such a complete evulsion; it is satisfactory if some 10 to 12 cm. of the nerve can be removed.

* Contribution to a discussion at the Medical Society of London, February 10th, 1930.